Modern Concepts of Cardiovascular Disease

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CHRONIC CONSTRICTIVE PERICARDITIS

The terminology in chronic pericardial disease remains confusing to many due to the existence of numerous synonyms and the use of broadly inclusive adjectives such as "adhesive" and "adherent." *Chronic constrictive pericarditis* (concretio cordis, Pick's disease) is a chronic disorder characterized by the presence of a diffusely scarred, thickened, often calcified pericardium, associated in almost all cases with total obliteration of the pericardial cavity. The condition is believed to be usually the result of tuberculous infection and may follow a tuberculous polyserositis. It results in splinting of the myocardium, a reduction in cardiac output, and symptoms and signs of chronic venous congestion. While the parietal pericardium may be joined by fibrous bands to adjacent tissues, such adhesions are not of great functional significance. *Chronic mediastino-pericarditis* (accretio cordis) is a term best reserved for that condition in which the pericardium is firmly attached by scar tissue to surrounding structures including the chest wall, diaphragm, pleura, mediastinum, and even vertebral column, thus rigidly fixing the heart in the thoracic cavity. The pericardial layers may be fused together and the pericardium somewhat thickened, but not to the degree and not with the splinting effect seen in constrictive pericarditis. It is considered to be the late result of rheumatic fever and is associated with valvular heart disease, which chiefly accounts for the unusual myocardial hypertrophy and disability present in these cases. *Polyserositis* (Concato's disease), believed to be usually tuberculous in origin, is often a precursor of constrictive pericarditis, and may be accompanied by pleuritis, peritonitis and by pericardial effusion with progressive inflammation and thickening of the pericardial layers. *Adhesive pericarditis* or an *adherent pericardium* are loose and unsatisfactory terms designating any condition in which the parietal and visceral pericardial surfaces are found to be fused. Obviously, chronic constrictive pericarditis and chronic mediastino-pericarditis fall in this group as do the pericardial obliterations (ordinarily of little if any significance) found following acute pericarditis of any type.

Chronic constrictive pericarditis is seen in males more than twice as often as in females, and affects all age groups, being most common between the ages of 10 and 40. In our experience, a history of exposure to tuberculosis is uncommon. It has been found that the three outstanding complaints are shortness of breath, swelling of the legs and abdominal swelling. Occasionally, a moderate degree of pericardial constriction may exist without significantly inconveniencing the patient. Distention of the neck veins, hepatomegaly, ascites, peripheral edema, and signs of pleural fluid are seen in more than half of the cases; a narrow pulse pressure and pulsus paradoxicus are frequent but less common and a pericardial friction rub is rare as might be expected from the nature of the disease. The heart may be normal in size, but, despite a common belief, may equally often be found to be moderately enlarged or rarely greatly enlarged.

Roentgen ray studies are helpful in disclosing calcification of the pericardium in approximately half of the patients, and about 85 per cent have been noted to show diminished cardiac pulsations on fluoroscopy. The electrocardiogram is also useful as flattening or inversion of the T waves is common in all leads, and low voltage and auricular fibrillation or flutter are frequently observed.

Lyons and Burwell[1] have shown that the crucial problem in chronic constrictive pericarditis is the limitation of diastolic filling of the ventricles, resulting in a diminished stroke volume and cardiac output, and an inability to increase the cardiac output on demand. It appears likely that not only are the ventricles limited in their capacity during diastole, but also they are restricted during systole in their contractile range and force as a result of the adherence of the myocardium to the rigid percardium. It has been suspected for some time that too much emphasis has been placed on the role of the right ventricle in this regard, and recent work[2, 3, 4] including data obtained by cardiac catheterization has shown that impaired function of the left ventricle is often of primary importance, being accompanied by elevation of the mean pulmonary "capillary", pulmonary arterial diastolic, and right ventricular diastolic pressures. In situations in which the clinical diagnosis is in doubt, cardiac catheterization may be useful in demonstrating not only these pressures with the characteristic narrow pulse pressure in the right ventricle, but also in showing the inability of the heart to increase its output under stress.

Pathological studies[2] have been confirmatory of the diffuse nature of the process, and it is clear that the left ventricle as well as the right ventricle and frequently both auricles are constricted, and that mild degenerative changes in the myocardium are not uncommon. Constricting rings around the venae cavae appear of minor significance. Pleuritis, commonly bilateral and often with effusion, is usually present at autopsy, and cardiac cirrhosis is found in advanced cases. In this regard, it has been observed that portal as well as central fibrosis exists, undoubtedly aggravated by the presence of intimal thickening and at times actual thrombosis of the hepatic veins, producing a mild Chiari syndrome.

It has long been surmised that tuberculosis was the etiological factor in most cases of chronic constrictive pericarditis, and new evidence in support of this concept has recently come from the studies of Andrews, Pickering and Sellors[5] in England. These workers have had an opportunity to follow the course of 16 cases of proved acute tuberculous pericarditis and have seen 14 of them develop chronic constrictive pericarditis. Edwards,[6] also in England, has had a similar experience with a smaller group of patients. The explanation for the rather low incidence of proved tuberculous cases reported in the American

literature may rest in part in the fact that most of the patients studied in this country were further along in their disease, and at the stage of healing or relative quiescence of any active tuberculous process in which the finding of tubercle bacilli or typical tubercle formation could not often be expected. The possibility of a differing mode or type of tuberculous infection in England and the United States cannot be entirely eliminated.

Surgical treatment with excision of large areas of the offending pericardium is indicated for virtually all patients with distressing or disabling symptoms who constitute good or even fair surgical risks. Patients with minimal impairment of cardiac function are best treated medically, reliance being placed on sodium restriction in the diet and the use of diuretics. In the light of the observations reported above, it is important when surgery is done that the approach be adequate to allow the operator to free the left ventricle from its constricting vise, and not merely the right auricle and ventricle. A left lateral incision may therefore be preferable to the classical anterior one. Even so, it is to be expected that certain patients will require more than one operation before satisfactory results are obtained (in this event, it is physiologically sound to release the left ventricle first).

Finally, Findley and Adams,[7] and members of our own group, have seen patients with primary amyloidosis involving the heart whose symptoms, physical findings, and laboratory studies so closely resembled those of chronic constrictive pericarditis as to make accurate diagnosis exceedingly difficult. There appears to be no single reliable differential diagnostic clue but a painstaking review of the features of any atypical case of presumed chronic constrictive pericarditis—especially one without pericardial calcification—may rarely lead to the detection of primary amyloid disease. Patients with myocarditis of unknown origin have occasionally presented similar diagnostic difficulties in the past.

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SCIENTIFIC SESSION

The Twenty-Fourth Scientific Session of the American Heart Association will be held at Chalfonte-Haddon Hall Hotel, Atlantic City, New Jersey, June 8 and 9, 1951. All those desiring to attend should make reservations directly with Hotels in Atlantic City immediately.

PROGRAM COMMITTEE

The Chairman of the Program Committee for the Annual Scientific Session is Doctor E. Cowles Andrus, 24 East Eager Street, Baltimore 2, Maryland. All who desire to present papers at the meetings in Atlantic City should forward to Doctor Andrus an abstract (in triplicate) of the proposed presentation of not more than 300 words. The deadline for the receipt of abstracts is February 10, 1951.

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